Appendix E4

Toxicity Parameters

TABLES

Table E4-1. Contaminant Specific Parameter Values

Contaminant of Potential Concern	Chemical Abstract Number	Region IX/III RBCs (mg/kg or pCi/g) [a]	Region	INEEL EBSLs (mg/kg or pCi/g) [d]	Oral RfD (mg/kg-day) [f]	Oral SF (mg/kg-day)$^{-1}$ or (risk/pCi) [f]	Inhalation RfD (mg/kg-day) [f]	Inhalation SF (mg/kg-day)$^{-1}$ or (risk/pCi)$^{-1}$ [f]
2,4,6-Trinitrotoluene	118-96-7	1.62E+01	9		5.00E-04	3.00E-02	5.00E-04	3.00E-02
2-Amino-4,6-Dinitrotoluene	35572-78-2	4.70E+00			5.00E-04	3.00E-02	5.00E-04	3.00E-02
2-Pentanone	107-87-9							
4-Amino-2,6-Dinitrotoluene	1946-51-0	4.70E+00			5.00E-04	3.00E-02	5.00E-04	3.00E-02
4-Chloro-3-methylphenol	59-50-7			1.80E+01				
Antimony	7440-36-0	3.13E+01	9	1.35E+00	4.00E-04			
Arsenic	7440-38-2	3.90E-01	9	8.44E-01	3.00E-04	1.50E+00		1.51E+01
Benzene	71-43-2	6.72E-01	9	5.50E+00	3.00E-03	2.90E-02	1.71E-03	2.70E-02
Benzo(a)pyrene	50-32-8	6.20E-02	9	2.69E+00		7.30E+00		3.10E+00
Benzo(g,h,i)perylene	191-24-2					7.30E-03		
Cadmium	7440-43-9	3.90E+01 [b]	3	2.36E-03	5.00E-04			6.30E+00
Copper	7440-50-8	2.90E+03	9	2.11E+00	3.71E-02			
Lead	7439-92-1	4.00E+02	9	9.94E-01				
Methapyrilene	91-80-5							
Phenanthrene [e]	85-01-8			1.35E+02		7.30E+00		3.10E+00
RDX	121-82-4	4.42E+00	9		3.00E-03	1.10E-01	3.00E-03	1.10E-01
Thallium	7440-28-0	5.48E+00	3	1.01E-01	7.00E-05			
TPH-Diesel	--	1.00E+03	[t]					
Cs-137	10045-97-3	2.30E-01	9	4.95E+03		3.16E-11		1.91E-11
Ra-226	13982-63-3	5.50E-03	9	2.04E+01		2.96E-10		2.75E-09
U-235	15117-96-1	1.30E-01	9	2.27E+01		4.52E-11		1.30E-08
U-238	7440-61-1	6.70E-01	9	2.32E+01		4.27E-11		1.24E-08

Table E4-1. (continued)

Contaminant of Potential Concern	External SF (risk/yr per pCi/g) [g]	Weight of Evidence [h]	Henry's Law Constant (atm-m^3/mol)	Kd (cm^3/g) [i]	Diffusivity (cm^2/s)	Absorption Factor [k]	Dermal Permeability (cm/h) [n]	Half-life (yr) [o]	MW (g/mol) [p]	PUF (mg/kg plant)/(mg/kg soil) [q]	Target Organ
2,4,6-Trinitrotoluene		C		5.30E+00 [s]		0.1 [l]	3.40E-03		2.27E+02	4.46E+00 [r]	
2-Amino-4,6-Dinitrotoluene						0.1	1.00E-03		1.97E+02		
2-Pentanone			1.24E+01			0.1	1.00E-03				
4-Amino-2,6-Dinitrotoluene						0.1	1.00E-03		1.97E+02		
4-Chloro-3-methylphenol			-		-	0.1	1.00E-03				
Antimony			-	5.00E+01	-	0.1	1.00E-03		1.22E+02	5.06E-04	whole body, blood;increased mortality
Arsenic		A	-	3.00E+00	-	3.00E-02	1.00E-03		7.49E+01	4.00E-02	skin;keratosis, hyperpigmentation
Benzene		A	5.55E-03	3.72E-01	8.8E-02	0.05	1.10E-01		7.8E+01	2.3E+00	
Benzo(a)pyrene		B2	1.55E-06	1.65E+04	4.30E-02	0.13	1.20E+00		2.52E+02	1.25E-02	
Benzo(g,h,i)perylene		D	5.34E-08	4.80E+03	4.20E-02	1.00E-01	1.62E+00		2.76E+02	3.05E-03	
Cadmium		B1	-	6.00E+00	-	0.001	1.00E-03		1.12E+02	5.50E-01	
Copper		D	-	2.00E+01	-	0.1	1.00E-03		6.35E+01	8.00E-01	gastrointestinal system;irritation
Lead		B2	--	1.00E+02		1.00E-01	1.00E-03		2.07E+02	2.00E-02	CNS;neurotoxicity, blood;toxicity
Methapyrilene						0.1	1.00E-03		2.61E+04		
Phenanthrene [e]		D	1.59E-04	4.23E+01	5.80E-02	3.00E-01	2.70E-01		1.78E+02	1.02E-01	
RDX		C		1.00E+00 [j]		0.01 [l]	1.00E-03		2.22E+02	1.03E+01 [r]	
Thallium			-	0.00E+00	-		1.00E-03		2.04E+02	4.00E-03	liver;increased SGOT, blood;increased serum LDH,

											hair;alopecia
TPH-Diesel		D	-	1.78E+00	-	0.1	6.90E-02				whole body;decreased body weight
Cs-137	2.09E-06	A	-	5.00E+02	-	0.95 [m]	1.00E-03	3.02E+01	1.37E+02	4.60E-01	
Ra-226	6.74E-06	A	--	1.00E+02		0.2 [m]	1.00E-03	1.60E+03	2.26E+02	1.00E-01	
U-235	2.63E-07	A	--	6.00E+00		0.05 [m]	1.00E-03	7.04E+08	2.35E+02	1.40E-02	
U-238	1.50E-11	A	--	6.00E+00		0.05 [m]	1.00E-03	4.47E+09	2.38E+02	1.40E-02	

Notes:

a) Risk based soil concentrations were taken from the EPA Region 9 Preliminary Remediation Goals presented on the EPA Region 9 website. If Region 9 didn't have values for the COPC, EPA Region 3 RBCs were used where available. Radionuclide RBCs were taken from Table 5 of the personnel communication from Jeff Fromm, Ph.D. an environmental toxicologist to the "INEL WAG Managers and Technical Support Staff." (1/3/96) "Radionuclide Risk-Based Concentration Tables."

b) Using the smaller of the Cadmium Water and Cadmium Food values presented in Region 3. From conversation with D. Burns 5/10/00.

c) Derived from I. Figueroa EDF on "Risk Analysis for determination of RBCs for Ordnanace Areas" (1992) and the US Army report on "Organic Explosives and Related Compounds: Environmental and health considerations." (1989)

d) INEEL EBSLs were taken from Appendix D of the Workplan for OU10-04 (1999) used the overall minimum EBSL value.

e) Slope factors and reference doses for Phenanthrene were taken from Benzo (a) pyrene, since they are in the same PAH family and Benzo (a) pyrene is more toxic.

f) Oral and inhalation slope factors and reference doses were taken from EPA Region 9 PRG tables. Which were summarized from the EPA's Integrated Risk Information System (IRIS), National Center for Environmental Assessment (NCEA) and the Health Effects Assesment Summary Tables (HEAST, 1997). If Region 9 numbers could not be found, Region 3 values were consulted.

g) External radiation slope factors were taken from the 1997 HEAST tables.

h) Weight of evidence values were taken from IRIS.

i) Kd's taken from INEEL Track 2 guidance (DOE-ID 1994), unless otherwise noted.

j) RDX's Kd was taken from the *Encyclopedia of Environmental Analysis and Remediation*, "Destruction of military toxic materials." Dr. R.W. Shaw and Dr. M. John Cullinane. Published by John Wiley & Sons, Inc.

k) Absorption factors were assigned a default value of 0.1 unless otherwise noted. Per Personnel communication (memo) from Bruce Becker to Doug Jorgensen, October 30. 1996, "Dermal Exposure Recommendations - BHB-07-96"

l) RDX and TNT's Absorption factors was taken from a personal communication with Carolyn Fordham a Toxicologist with Earthtech.m) Absoprtion factors for the radionuclides were taken from HEAST 1997.

n) Dermal permeability values were assigned the default value of 0.001 unless otherwise noted. o) Half lives were taken from HEAST 1997

p) Molecular weights were taken from the *Dictionary of Chemical Names and Synonyms*. Philip H. Howard and Michael Neal, 1992, Lewis Publisheres.

q) Plant uptake factors were taken from the INEEL *White paper on Food Crop Ingestion Exposure Route* (LMITCO 1996), unless otherwise noted.

r) TNT and RDX's PUFs were taken from site specific data collected at Tooele Army depot (Rust Environment and Infrastructure, 3/98)

s) TNT's Kd was taken from "Adsorption and Desportion of 2,4,6-TNT by Soils." Judith C. Pennington and William H. Patrick, Jr. Journal of Environmental Quality. 19:559-567 (1990)

t) TPH-Diesel RBC levels were taken from a personnel communication from G.C Bowman, Director of the DOE-ID Environmental Protection Division, November 29, 1989

u) Oral and Inhalation SFs and RfD's for 2-Amino-4,6-Dinitrotoluene and 4-Amino-2,6-Dinitrotoluene were taken from TNT values listed in IRIS, per Carolyn Fordham, an environmental toxicologist with Earthtech.

Appendix E5

Toxicity Profiles

CONTENTS

Appendix E5

Toxicity Profile

E5-1 TOXICITY PROFILE - 1,3,5- TRINITROBENZENE

E5-1.1.1 Chemical Properties and Fate

1,3,5-Trinitrobenzene (TNB) is a co-contaminant of trinitrotoluene (TNT) production (Layton et al. 1987). TNB is a photolytic breakdown product in addition to being an impurity. Data suggest that TNB can undergo biotransformation (Layton et al. 1987). TNB occurs where TNT has been a surface soil or surface water contaminant for some time (Burrows et al. 1989).

The vapor pressure is reported as 1E-4 torr (Layton et al. 1987). A log Kow of 1.18 was measured (Layton et al. 1987). A log Koc of 1.88 was estimated. The water solubility is 330 mg/L (0.0015 mol/L) at a temperature of 20°C (Layton et al. 1987). The gram molecular weight (MW) is 213.1.

E5-1.1.2 Bioaccumulation

Plant-soil partition coefficients are useful in predicting tissue concentrations in plants that could be used as food for human or ecological receptors. Plant concentrations change over time in response to differences in uptake or loss, but generally uptake is higher earlier in the growing cycle, and slows as the plant matures (Layton et al. 1987). Plant concentration factors (Ksp), which fall within the range estimated by Small (1984) for different classes of organic chemicals, were estimated by the following equation from Topp et al. (1986), which is based on measured uptake values for barley for 14 chemicals and had a r^2 of 0.89:

$$\log Ksp = 5.943 - 2.385 * \log MW$$

The predicted uptake factor or Ksp for plants is 2 (Layton et al. 1987).

Measured data for uptake by fish or other aquatic life were unavailable in the literature reviewed. Concentrations in fish were predicted with the following equation from Vieth and Kosian (1983) (Layton et al. 1987). This equation estimates a bioconcentration factor (BCF) for fish, and was developed from data for 122 organic chemicals and has an r^2 of 0.86:

$$\log Kfw = 0.79 * \log Kow - 0.40$$

The predicted concentration factor (Kfw) in fish, based on the log Kow of 1.18, was 3.4.

Estimation of the concentration in beef fat can also be made from regression equations developed by Kenaga (1980) (Layton et al. 1987). These equations relate the Kow and aqueous solubility (S) in mg/L of organic chemicals to their partitioning between diet and fat of cattle. Based on 28 day feeding trials with 23 organic chemicals, the equations are as follows:

$$Log\ Kfd = 0.5 * Log\ Kow - 3.457 \quad (n = 23; r^2 = 0.62)$$

$$Log\ Kfd = -0.495 * Log\ S - 1.476 \quad (n = 23, r^2 = 0.67)$$

The estimated Kfd is 1.4E-3 based on a log Kow of 1.18, and the estimated Kfd based on water solubility was 1.9E-3 based on a water solubility of 330 mg/L. These values indicate that unless dietary concentrations are extremely high, grazing mammals should not contain large amounts of TNB. This is further supported by tests that indicate that 10% of a dose is eliminated in urine within 24 hours, and up to 40% of the dose is eliminated within four days in urine and feces combined (Reddy et al. 1997). Low levels remained in tissue after four days.

E5-1.1.3 Aquatic Toxicity

The measured LC50 for fathead minnows (*Pimephales promeleus*) is 1 mg/L, whereas an LC50, predicted on the basis of structure activity relationships, is 7.9 mg/L (Gao et al. 1992). Burrows (1989) reports 96-h LC50 for the fathead minnow of 0.5 to 1.1 mg/L and 48-h LC50 for the water flea (*Daphnia magna*) of 2.7 to 3.0 mg/L.

E5-1.1.4 Terrestrial Toxicity

A literature search was conducted with the TOXLINE bibliographic data base August 2000. No information was obtained in the literature reviewed.

E5-1.1.5 Avian Toxicity

A literature search was conducted with the TOXLINE bibliographic data base August 2000. No information was obtained in the literature reviewed.

E5-1.1.6 Mammalian Toxicity

TNB is not a dermal irritant and does not produce dermal toxicity at concentrations up to 2 g/kg body weight (bw), but is an eye irritant and can produce mild skin sensitization (Reddy et al. 1997). Toxic effects are characterized by respiratory disorders, cyanosis, and central nervous system effects (Reddy et al. 1997).

FitzGerald et al. (1992) conducted acute oral toxicity tests with rodents. Tests with rats produced LD50 values of 298, 275, and 284 mg/kg for male, female and combined sexes. Tests with mice yielded LD50 values of >900 mg/kg for male mice and 702 mg/kg for female mice. Acute toxicity to rodents, as indicated by an oral LD50, was reported by Burrows et al. (1989). The LD50 for rats was 450 mg/kg, and the LD50 for mice was 572 mg/kg.

Subchronic (90-d) oral toxicity tests were conducted with rats fed 0, 66.67, 400, and 800 mg TNB/kg diet, where intakes for males were 0, 4.3, 24.7, and 49.3 mg/kg bw/d, and daily intakes for females were somewhat less (Reddy et al. 1994a, 1994b). Rats receiving a 400 and 800 mg/kg diet consumed less food resulting in a decrease in body weight. Water consumption in the high dose female rats and relative organ weights (g organ/g bw) in both sexes was increased significantly, and relative testicular weight was decreased significantly in males (Reddy et al. 1997). Exposure resulted in hematological effects, such as a decrease in total red cell count and hemoglobin content, and an increase in methemoglobin in the 400 and 800 mg/kg treatment groups. Histopathological analysis indicated moderate to severe seminiferous tubular degeneration in testis of the 400 and 800 mg/kg treatment groups. The spleen and bone marrow had mild to moderate effects in rats of both sexes in the 400 and 800 mg/kg treatments. A No Observed Adverse Effect Level (NOAEL) of 4.3 mg/kg bw/d was established for female rats, and no NOAEL can be suggested for male rats since toxic effects in the male kidney were observed at all doses tested. A Lowest Observed Adverse Effect Level (LOAEL) of 3.9 mg/kg bw/d is recommended by the author.

Subchronic (90 d) toxicity was evaluated in the white-footed mouse (*Peromyscus leucopus*), where animals of both sexes were fed diets containing 0, 150, 375, and 750 mg/kg diet for an average estimated daily consumption of TNB of 0, 23.50, 67.44, and 113.51 mg/kg bw/d for male mice and slightly less for females (Reddy et al. 1995). The only significant biological findings were in the 750 mg/kg treatment group. Significant dose-related increases in relative organ weights, histopathological changes in spleen (erythroid cell hyperplasia), and testis (seminiferous tubule degeneration) were observed in this group. A NOAEL of 20.1 mg/kg bw/d was suggested for female mice, and 23.5 mg/kg bw/d for male mice.

Rats were tested for reproductive effects of TNB had no adverse effects on reproductive indices when fed a diet of 30, 150, and 300 mg of TNB/kg diet (3, 14, and 29 mg/kg bw/d for females) (Kinkead et al. 1994, 1995). Rats were fed TNB 14 days prior to mating, and for four weeks postweaning for a total of a 90 day exposure. No mortality occurred in the parental animals. Absolute and relative organ weights were affected at the 300 mg/kg treatment, sperm effects were observed in animals dosed with this treatment as well. No significant dose-related effects were observed in length of gestation, sex ratio, or mean number of offspring per litter. A NOAEL based on reproductive toxicity endpoints including mating, fertility, and others were 2 mg/kg bw/d for males and 3 mg/kg bw/d for females. A NOAEL for developmental toxicity was 45 mg/kg bw/d.

E5-1.1.7 Human Toxicity

Not requested.

E5-1.1.8 Recommendations

The lowest NOAEL for mammals should be used to establish quantitative risk estimates for mammalian receptors. The lowest aquatic LC50 divided by 100 or 1000 should be used to establish quantitative risk estimates for aquatic life. Data are inadequate to make quantitative risk estimates for other receptor groups.

E5-2 TOXICITY PROFILE
2-AMINO-4,6-DINITROTOLUENE

E5-2.1.1 Chemical Properties and Fate

2-Amino-4,6-dinitrotoluene (2-amino-4,6-DNT) is a biotransformation product of TNT (Layton et al. 1987). Aminodinitrotoluenes are produced by microbial action, where one or more of the NO_2 groups on the toluene molecule are reduced to NH_2 groups (Layton et al. 1987). Microbes reduce TNT faster under aerobic than anerobic conditions, and most of the products are mono- and diamines (Layton et al. 1987). Less TNT or its biotransformation products can be extracted from organic-rich sediments and soils than would be predicted on the basis of calculated or measured partition coefficients, suggesting microbial reduction ultimately produces insoluble precipitates (Layton et al. 1987).

The structure of the TNT metabolites determines the rate of photocatalytic degradation. For the monaminodinitrotoluenes, compounds with an amino group para to the methyl group degrade more rapidly than those with an ortho amino group (Schmidt and Butte 1999). 2-Amino-4,6-DNT is one of the major urinary metabolites of TNT in humans (Layton et al. 1987).

A log Kow of 0.5 was estimated (Layton et al. 1987). A log Koc of 0.15 was also estimated. The water solubility is 2800 mg/L (0.014 mol/L) at a temperature of 20 to 25°C (Layton et al. 1987). The gram molecular weight (MW) is 197.1.

E5-2.1.2 Bioaccumulation

Plants can absorb TNT and produce aminodinitrotoluene biotransformation products. Yellow nutsedge (*Cyperus esculentus*) grown in hydroponic media containing 5 to 20 mg/L TNT solutions for 42 d contained 2-amino-4,6-DNT and 4-amino-2,6-dinitrotoluene (4-amino-2,6 DNT). Metabolite concentrations in roots were up to 18 times that of the parent compound (Palazzo and Leggett 1986a). The tuber also showed a similar pattern of absorption and distribution, but to a lesser extent. Poplar trees retained 75% of TNT in their roots, and transformed TNT to 2-amino-4,6-DNT and 4-amino-2,6-DNT (Thompson et al. 1998).

Plant-soil partition coefficients are useful in predicting tissue concentrations in plants that could be used as food for human or ecological receptors. Plant concentrations change over time in response to differences in uptake or loss, but generally uptake is higher earlier in the growing cycle and slows as the plant matures (Layton et al. 1987). Plant concentration factors (Ksp), which fall within the range estimated by Small (1984) for different classes of organic chemicals, were estimated by the following equation from Topp et al. (1986), which was based on measured uptake values for 14 chemicals and had a r^2 of 0.89:

$$\log Ksp = 5.943 - 2.385 * \log MW$$

The predicted uptake factor or Ksp for plants, specifically barley, for 2-amino-4,6-DNT is 3 (Layton et al. 1987).

Measured data for uptake by fish or other aquatic life were unavailable in the literature reviewed. Concentrations in fish were predicted with the following equation from Vieth and Kosian (1983) (Layton et al. 1987). This equation estimates a bioconcentration factor (BCF) for fish, and was developed from data for 122 organic chemicals and has an r^2 of 0.86:

$$\log Kfw = 0.79 * \log Kow - 0.40$$

The predicted concentration factor (Kfw) in fish, based on the log Kow of 0.5, was 1 (Layton et al. 1987).

Estimation of the concentration of 2-amino-4,6-DNT in beef fat can also be made from regression equations developed by Kenaga (1980) (Layton et al. 1987). These equations relate the Kow and aqueous solubility (S) in mg/L of organic chemicals to their partitioning between diet and fat of cattle. Based on 28-day feeding trials with 23 organic chemicals, the equations are as follows:

$$Log\ Kfd = 0.5 * Log\ Kow - 3.457 \quad (n = 23; r^2 = 0.62)$$

$$Log\ Kfd = -0.495 * Log\ S - 1.476 \quad (n = 23, r^2 = 0.67).$$

The estimated Kfd is 6.2E-4 based on a log Kow of 0.5, and the estimated Kfd based on water solubility was 6.57E-4 based on a water solubility of 2800 mg/L. These values indicate that unless dietary concentrations of 2-amino-4,6-DNT are extremely high, or dietary concentrations of TNT are high, resulting in uptake and transformation of TNT to 2-amino-4,6-DNT, grazing mammals should not contain large amounts of 2-amino-4,6-DNT.

E5-2.1.3 Aquatic Toxicity

The measured LC50 for fathead minnows is 15 mg/L, whereas a LC50 predicted on the basis of structure activity relationships is 41 mg/L (Gao et al. 1992). 2-amino-4,6-DNT is considered toxic to aquatic organisms (Drzyzga et al. 1995).

E5-2.1.4 Terrestrial Toxicity

A literature search was conducted with the TOXLINE bibliographic data base August 2000. This literature search obtained one paper regarding the toxicity of TNT and its metabolites in plants (Palazzo and Leggett 1986b). TNT at solutions of 0.5 and 5 mg/L produced changes in physiological activity, where new plant growth became inhibited. Growth of roots, rhizomes, and leaves was inhibited. TNT and metabolites were found throughout the plant. Thus, toxicity cannot be separated from exposure to the parent compound.

E5-2.1.5 Avian Toxicity

A literature search was conducted with the TOXLINE bibliographic data base August 2000. No information was obtained in the literature reviewed.

E5-2.1.6 Mammalian Toxicity

A literature search was conducted with the TOXLINE bibliographic data base August 2000. No information was obtained in the literature reviewed.

E5-2.1.7 Human Toxicity

Urine of exposed workers was tested for mutagenic activity (Ahlborg et al. 1988). While correlations between the concentrations of urinary TNT and 2-Amino-4,6-DNT and 4-Amino-2,6-DNT were strong, the correlations between urine mutagenicity and TNT, 2-Amino-4,6-DNT, and 4-Amino-2,6-DNT concentrations were weak, and statistically significant only for 4-Amino-2,6-DNT. Other studies also suggest that TNT metabolites are mutagenic (Brooks et al. 1998). 2-Amino-4,6-DNT had in vitro cytotoxicity similar to TNT with cellular LC50s in the 3 to 18 ug/ml range (Honeycutt et al. 1996).

Tan et al. (1992) report that mutagenicity decreased with increasing nitro groups reduced to the amino form; thus, the metabolites would be less mutagenic than the parent TNT compound.

E5-2.1.8 Recommendations

The toxicity values for human and ecological receptors for TNT should be used to estimate risks to the metabolites. The data are too limited to establish any type of human or ecological toxicological criteria, although the data appear to suggest that toxicity is less than or similar to that of TNT.

E5-3 TOXICITY PROFILE
2-PENTANONE (METHYL-N-PROPYL KETONE)

E5-3.1.1 Chemical Properties and Fate

Ketones can potentiate the acute and chronic hepatotoxic effects of haloalkanes such as carbon tetrachloride and chloroform (Plaa 1988).

The partition coefficient between water and air is 166. The Log Kow is 2.80 (Kumagai et al. 1999). The molecular weight is 86.13.

E5-3.1.2 Bioaccumulation

Plant-soil partition coefficients are useful in predicting tissue concentrations in plants that could be used as food for human or ecological receptors. Plant concentrations change over time in response to differences in uptake or loss, but in general uptake is higher earlier in the growing cycle and slows as the plant matures (Layton et al. 1987). Plant concentration factors (Ksp), which fall within the range estimated by Small (1984) for different classes of organic chemicals, were estimated by the following equation from Topp et al. (1986), which is based on measured uptake values for barley for 14 chemicals and had a r^2 of 0.89:

$$\log Ksp = 5.943 - 2.385 * \log MW$$

The predicted uptake factor or log Ksp for plants is 1.3, which yields a Ksp of 20 (Layton et al. 1987).

Measured data for uptake by fish or other aquatic life were unavailable in the literature reviewed. Concentrations in fish were predicted with the following equation from Vieth and Kosian (1983) (Layton et al. 1987). This equation estimates a bioconcentration factor (BCF) for fish, and was developed from data for 122 organic chemicals and has an r^2 of 0.86:

$$\log Kfw = 0.79 * \log Kow - 0.40$$

The predicted concentration factor (Kfw) in fish, based on the log Kow of 2.80, was 64.9.

Estimation of the concentration in beef fat can also be made from regression equations developed by Kenaga (1980) (Layton et al. 1987). These equations relate the Kow to the partitioning between diet and fat of cattle. Based on 28 day feeding trials with 23 organic chemicals, the equations are as follows:

$$Log\ Kfd = 0.5 * Log\ Kow - 3.457 \quad (n = 23;\ r^2 = 0.62)$$

The estimated Log Kfd is –2.06 based on a Log Kow of 2.80, which results in a Kfd of 0.0088 after taking the antilog.

E5-3.1.3 Aquatic Toxicity

No information was available in the literature reviewed.

E5-3.1.4 Terrestrial Toxicity

No information was available in the literature reviewed.

E5-3.1.5 Mammalian Toxicity

Rats administered a single oral dose of 15 mmol/kg exhibited no mortality, and urinary tract endpoints indicated no significant change from controls (Hewitt and Brown 1984). Guinea pigs were exposed to 2-pentanone in the air for up to 810 minutes (Yant et al. 1936). The no effect level was 0.15%. An air concentration of 0.5% produced dyspnea, gasping, and unconsciousness; whereas, a concentration of 0.8% and higher caused mortality. In mice, the effective concentration producing an effect in 50% of the test subjects (EC50) was 5,915 ppm in air for respiratory effects and 1,348 ppm for neurobehavioral effects (De Ceaurriz et al. 1984).

E5-3.1.6 Human Toxicity

Air concentrations as low as 0.15% produce a disagreeable odor, which is irritating to the eyes and nose (Yant et al. 1936). Two ketones with a structure similar to 2-pentanone, methyl butyl ketone and methyl ethyl ketone have been linked to motor neuropathy in an occupational setting (Allen et al. 1974). Other data suggest that methyl propyl ketone is not neurotoxic (Misumi and Nagano 1984).

E5-3.1.7 Recommendations

Sufficient data for making quantitative risk estimates are lacking for this chemical. One solution is to use the air concentration of 0.15% (1,500 ppm) as a lower effect level for air. Soil or surface water concentrations that would result in air concentrations below this value could then be estimated with equations using the Henry's Law constant and other appropriate parameters.

E5-4 TOXICITY PROFILE
4-AMINO-2,6-DINITROTOLUENE

E5-4.1.1 Chemical Properties and Fate

4-Amino-2,6-dinitrotoluene (4-amino-2,6-DNT) is a biotransformation product of TNT (Layton et al. 1987). Aminodinitrotoluenes are produced by microbial action, where one or more of the NO_2 groups on the toluene molecule are reduced to NH_2 groups (Layton et al. 1987). Microbes reduce TNT faster under aerobic than anerobic conditions, and most of the products are mono- and diamines (Layton et al. 1987). Less TNT or its biotransformation products can be extracted from organic-rich sediments and soils than would be predicted on the basis of calculated or measured partition coefficients, suggesting microbial reduction ultimately produces insoluble precipitates (Layton et al. 1987).

The structure of the TNT metabolites determines the rate of photocatalytic degradation. For the monaminodinitrotoluenes, compounds with an amino group para to the methyl group degrade more rapidly than those with an ortho amino group (Schmidt and Butte 1999). 4-Amino-2,6-DNT is one of the major urinary metabolites of TNT in humans (Layton et al. 1987).

A log Kow of 0.6 was estimated (Layton et al. 1987). A log Koc of 0.25 was also estimated. The water solubility is 2,800 mg/L (0.014 mol/L) at a temperature of 20 to 25°C (Layton et al. 1987). The gram molecular weight (MW) is 197.1.

E5-4.1.2 Bioaccumulation

Plants can absorb TNT and produce aminodinitrotoluene biotransformation products. Yellow nutsedge (*Cyperus esculentus*) grown in hydroponic media containing 5 to 20 mg/L TNT solutions for 42 d contained 2-amino-4,6-DNT and 4-amino-2,6 DNT. Metabolite concentrations in roots were up to 18 times that of the parent compound (Palazzo and Leggett 1986a). The tuber also showed a similar pattern of absorption and distribution, but to a lesser extent. Poplar trees retained 75% of TNT in their roots and transformed TNT to 2-amino-4,6-DNT and 4-amino-2,6-DNT (Thompson et al. 1998).

Plant-soil partition coefficients are useful in predicting tissue concentrations in plants that could be used as food for human or ecological receptors. Plant concentrations change over time in response to differences in uptake or loss, but generally uptake is higher earlier in the growing cycle, and slows as the plant matures (Layton et al. 1987). Plant concentration factors (Ksp), which fall within the range estimated by Small (1984) for different classes of organic chemicals, were estimated by the following equation from Topp et al. (1986), which was based on measured uptake values for 14 chemicals and had a r^2 of 0.89:

$$\log Ksp = 5.943 - 2.385 * \log MW$$

The predicted uptake factor or Ksp for plants, specifically barley, for 4-amino-2,6-DNT is 3 (Layton et al. 1987).

Measured data for uptake by fish or other aquatic life were unavailable in the literature reviewed. Concentrations in fish were predicted with the following equation from Vieth and Kosian (1983) (Layton et al. 1987). This equation estimates a bioconcentration factor (BCF) for fish, and was developed from data for 122 organic chemicals and has an r^2 of 0.86:

$$\log Kfw = 0.79 * \log Kow - 0.40$$

The predicted concentration factor (Kfw) in fish, based on the log Kow of 0.6, was 1 (Layton et al. 1987).

Estimation of the concentration of 4-amino-2,6-DNT in beef fat can also be made from regression equations developed by Kenaga (1980) (Layton et al. 1987). These equations relate the Kow and aqueous solubility (S) in mg/L of organic chemicals to their partitioning between diet and fat of cattle. Based on 28 day feeding trials with 23 organic chemicals, the equations are as follows:

$$Log\ Kfd = 0.5 * Log\ Kow - 3.457 \quad (n = 23; r^2 = 0.62)$$

$$Log\ Kfd = -0.495 * Log\ S - 1.476 \quad (n = 23, r^2 = 0.67)$$

The estimated Kfd is 7.0E-4 based on a log Kow of 0.5, and the estimated Kfd based on water solubility was 6.57E-4 based on a water solubility of 2800 mg/L. These values indicate that unless dietary concentrations of 4-amino-2,6-DNT are extremely high or dietary concentrations of TNT are high, resulting in uptake and transformation of TNT to 4-amino-2,6-DNT, grazing mammals should not contain large amounts of 4-amino-2,6-DNT.

E5-4.1.3 Aquatic Toxicity

4-amino-2,6-DNT is considered toxic to aquatic organisms (Drzyzga et al. 1995). The measured LC50 for fathead minnows is 6.84 mg/L, whereas an LC50 predicted on the basis of structure activity relationships is 41 mg/L (Gao et al. 1992).

E5-4.1.4 Terrestrial Toxicity

A literature search was conducted with the TOXLINE bibliographic data base August 2000. This literature search obtained one paper regarding the toxicity of TNT and its metabolites in plants (Palazzo and Leggett 1986b). TNT at solutions of 0.5 and 5 mg/L produced changes in physiological activity, where new plant growth became inhibited. Growth of roots, rhizomes, and leaves was inhibited. TNT and metabolites were found throughout the plant. Thus, toxicity cannot be separated from exposure to the parent compound.

E5-4.1.5 Avian Toxicity

A literature search was conducted with the TOXLINE bibliographic data base August 2000. No information was obtained in the literature reviewed.

E5-4.1.6 Mammalian Toxicity

A literature search was conducted with the TOXLINE bibliographic data base August 2000. No information was obtained in the literature reviewed.

E5-4.1.7 Human Toxicity

Urine of exposed workers was tested for mutagenic activity (Ahlborg et al. 1988). While correlations between urinary concentrations of TNT, 2-Amino-4,6-DNT, and 4-Amino-2,6-DNT were strong, the correlations between urine mutagenicity and TNT, 2-Amino-4,6-DNT, and 4-Amino-2,6-DNT concentrations were weak and statistically significant only for 4-Amino-2,6-DNT. Other studies also suggest that TNT metabolites are mutagenic (Brooks et al. 1998). 4-Amino-2,6-DNT was generally less cytotoxic than TNT in *in vitro* studies (Honeycutt et al. 1996). Tan et al. (1992) report that mutagenicity

decreased with increasing nitro groups reduced to the amino form; thus, the metabolites would be less mutagenic than the parent TNT compound.

E5-4.1.8 Recommendations

The toxicity values for human and ecological receptors for TNT should be used to estimate risks to the metabolites. The data are too limited to establish any type of human or ecological toxicological criteria, although the data appear to suggest that toxicity is less than or similar to that of TNT.

E5-5 TOXICITY PROFILE
CHLORIDE

E5-5.1.1 Chemical Properties and Fate

Most of the chlorine on earth exists as chloride ion (Cl^-), which is the predominant ion in seawater on a mass and molar basis (Bodek et al. 1988). The chloride concentration in seawater is 19,350 mg/L (0.55M), the abundance of which is explained by the chemistry of chlorine versus chloride. Chlorine is highly volatile. It separated from crustal rock early in the geochemical history of the earth and entered the atmosphere. Chlorides are highly soluble in water, and once dissolved in rainwater end up in the oceans. Chloride in the atmosphere washes out, and also eventually migrates to the oceans. Chloride moves through soils at the same rate as water, with no adsorption or retention (Bodek et al. 1988).

Chloride is essential for mammals and probably all organisms (Bowen 1979).

E5-5.1.2 Bioaccumulation

No information was obtained in the literature reviewed.

E5-5.1.3 Aquatic Toxicity

Freshwater organisms are not adapted to high levels of salts in water. Seawater is considered to be 3.5% total salts, estuarine water is 0.05 to 3.0% salts, and freshwater is <0.05% salts (Rand et al. 1995). Very hard freshwater contains 16 mg/L KCl (ASTM 1996). Therefore, if chloride concentrations remain within the normal values for freshwater, toxicity to aquatic life is not expected.

E5-5.1.4 Terrestrial Toxicity

Chloride and other salts can produce osmotic stress in plants (Bodek et al. 1988). High salt concentrations in soil make it difficult for plants to take up water. In plants that are not adapted for a saline environment, chloride toxicity occurs as high concentrations near the end of the plant's transpiration stream. This leads to necrosis and burning of leaf tips and margins, and to plant death. Only high chloride levels, such as those that result from deicing of roads by applying salt, are expected to produce toxicity to plants.

E5-5.1.5 Mammalian Toxicity

Chloride is not considered toxic except at very high concentrations (Bodek et al. 1988). The chloride ion is not chemically similar to other, more oxidized forms of chlorine, such as aqueous chlorine (Cl_2), hypochlorous acid (HOCl), or the hypochlorite ion (OCl^-), all of which are highly toxic. Chloride can affect the behavior of heavy metal cations; however, toxicity and uptake is associated with the free metal concentration or the heavy metal species (Bodek et al. 1988).

E5-5.1.6 Human Toxicity

The World Health Organization (WHO) recommended maximum concentration in drinking water is 250 mg/L based on the taste imparted to water when chloride concentrations exceed 200 to 300 mg/L (Bodek et al. 1988). This indicates that at a consumption rate of 2 L per day, over 600 mg of chloride can be safely consumed daily in drinking water alone. For a 70 kilogram human, this equates to a safe consumption rate of over 8.6 mg/kg bw/d. Safe doses could be even higher than this, since the drinking water concentration is based on taste and not a health effect.

E5-5.1.7 Recommendations

Only extremely high chloride concentrations will be toxic. Therefore, it is recommended that chloride not be considered further as a contaminant of potential concern.

E5-6 TOXICITY PROFILE
FLUORIDE

E5-6.1.1 Chemical Properties and Fate

Fluorine makes up 0.06 to 0.09% of the lithosphere, and is widely distributed in both the lithosphere and the hydroshpere (Bodek et al. 1988). The fluoride ion is the predominant form of fluorine under natural conditions. It is probably an essential element for animals, but at high concentrations it is toxic to both plants and animals (Bodek et al. 1988). Many industries release fluorine into the atmosphere, and fluorides in windblown dust and emissions from volcanic activity also contribute to the atmospheric load (Bodek et al. 1988).

Fluoride is added to soils as an impurity of phosphate fertilizers, which contain fluoride at levels of 0.5 to 4% by weight (Bodek et al. 1988). Fluorapatite is the major constituent of the phosphate rock used in the manufacture of phosphate fertilizers. Fluoride also enters soils through atmospheric deposition (Bodek et al. 1988). Over 90% of natural fluorides in soil are insoluble or bound to soil particles; however, surface concentrations tend to be lower than concentrations at depth, suggesting soluble fluorides are leached from surficial soils (Bodek et al. 1988).

E5-6.1.2 Bioaccumulation

Plants can bioaccumulate fluoride from the air. Deposition of gaseous and particulate fluorides leads to plant concentrations that are toxic to livestock (Bodek et al. 1988). Results regarding uptake from soils when fluoride is added in fertilization or pollution are extremely variable. This may be due to soil characteristics. Fluoride is more available from high-clay soils than sandy soils, and the presence of calcium decreases availability (Bodek et al. 1988). The effect of soil pH on bioavailability is conflicting and unclear with some studies suggesting increased soil sorption with decreasing pH, while other studies report increased availability with decreasing pH (Bodek et al. 1988).

Estimated bioaccumulation factors (BAFs) for terrestrial invertebrates range from 0.4 to 1.3 (Port et al. 1998). Estimated BAFs for mammals ranged from 0.25 to 0.41 (Boulton et al. 1994).

About 99% of the body burden in animals is in the bones and teeth (CEPA 1993); thus, higher level predators should not be highly exposed to fluorides in prey. Herbivores are the receptor group most likely at risk due to dietary fluoride exposure.

E5-6.1.3 Aquatic Toxicity

Lethal and nonlethal effects are observed in aquatic invertebrates, fish, and amphibians at concentrations above 3 mg/L (CEPA 1993). Fluoride toxicity is negatively correlated to water hardness and positively correlated to water temperature (CEPA 1993).

E5-6.1.4 Terrestrial Toxicity

Extensive information is available for toxicity to plants due to fluorides in the air (CEPA 1993), but less information is available for soils. Since fertilizer added as an amendment to soil can contain up to 0.5 to 4% fluorides by weight (500 to 4,000 mg/kg), soil concentrations one or two orders of magnitude below this should not be toxic to plants. Thus, a soil concentration unlikely to adversely affect plants is estimated to be 5 to 50 mg/kg.

E5-6.1.5 Avian Toxicity

Fluoride is not particularly toxic to birds. Screech owls (*Otus asio*) fed 200 mg/kg diet had lower reproductive success, but no effects were observed for birds fed 40 mg/kg diet (Pattee et al. 1988; Hoffman et al. 1985).

E5-6.1.6 Mammalian Toxicity

Effects in livestock are similar to those observed for humans (Bodek et al. 1988). Forage containing fluoride concentrations of 30 to 40 mg/kg is extremely toxic to cattle (Bodek et al. 1988). Other livestock species do not seem to be as sensitive. Deer exposed to 35 mg/kg fluoride in diet had mottling of teeth (CEPA 1993). Up to 5 mg/kg fluoride can be considered background levels in dietary items including meat, fish, and eggs (CEPA 1993).

Female rats dosed in drinking water for 20 days (throughout gestation) with 0, 10, 25, 100, 175, or 250 ppm (0, 1.4, 3.9, 15.6, 24.7, 25.1 mg/kg bw-d) NaF daily had no behavior or clinical signs (Collins et al. 1995). Decreased food intake and body weight by 12% was observed in animals dosed with 250 ppm. Decreased water intake was observed at 175 ppm. There were no effects on reproductive endpoints, including the viable fetuses and fetal development or weight. A NOAEL for this study for fluoride for rats would be 100 mg/L or 15.6 mg/kg-d.

Other studies report no effect levels of 12.7 mg/kg bw/d (for bone mineralization effects in rats); however, lowest observed effect levels much lower than this have been reported. Effects were reported at 4.7 mg/kg bw/d in rats administered water containing fluoride at concentrations of 8.5 mg/L for 21 days, and chronic effects were reported at 3.2 mg/kg bw/d for fluoride administered in water to rats at concentrations of 36.3 mg/L for 250 days (CEPA 1993). Bank voles (*Clethrionomys glareolus*) had reproductive effects when fed a diet of 97 mg/kg fluoride (Krasowska 1989).

E5-6.1.7 Human Toxicity

Fluoride is added to drinking water to prevent cavities; the concentration as a public health measure is 1 mg/L (Bodek et al. 1988). At concentrations >2 mg/L, mottling of teeth can occur, and at concentrations of 3 to 6 mg/L, skeletal fluorosis may be observed (Bodek et al. 1988).

E5-6.1.8 Recommendations

Unless fluoride is found in soils or biota at concentrations above 5 mg/kg, it does not appear that adverse effects are likely. Concentrations below 2 mg/L in surface water should be adequately protective of drinking water for grazing mammals or livestock. This would also be protective of aquatic life.

E5-7 FATE AND TRANSPORT/BACKGROUND LEVELS OF FLUORIDE IN SOIL

E5-7.1.1 Fate of Fluoride in Soil

Over 90% of the natural fluoride content of soils is insoluble or tightly bound to soil particles. Soils tend to have lower concentrations of fluoride at their surface than at a depth of a few feet, indicating that water seeping into the ground may remove soluble fluoride from the surface and that little fluoride is available for uptake by plants; however, research results vary concerning the degree to which fluoride added by pollution or fertilization is available for uptake by plant roots. Several soil characteristics influence the availability of fluoride. It is more readily available in high-clay soils than in sandy ones, and calcium tends to increase F^- immobilization (Bodek 1988).

E5-7.1.2 Background Levels of Fluoride Found in Soil

The typical soil composition for fluoride has a range of 20 to 700 mg/kg with a median concentration of 200 (Bowen 1979). Moreover, in a more comprehensive study conducted on numerous soil types, it was discovered that the levels of fluoride ranged from 10 to 1,900 mg/kg with a mean concentration of 280 mg/kg (Shacklette and Boerngen 1984). The background concentration of elements in soil differs based on the parent material and genesis of the soil, increasing the importance of comparing background levels from similar soil types. Most of the sites that were sampled for flouride are found on the flood plain. The flood plain soils were derived from alluvial deposits of the Big Lost River and Birch Creek (Olson et al. 1995). The background levels of fluoride found in alluvial soils ranges from 10 to 1200 mg/kg with a mean concentration of 465 mg/kg (Kabata-Pendias 1985). The maximum fluoride contents of INEEL soils, from the Operable Unit (OU) 10-04 sites, ranged from 130 to 340 mg/kg. However, only two samples were found at this upper range (340 and 300 mg/kg); the next highest sample for fluoride was 240 mg/kg. These concentrations are comparable to the background levels of fluoride in the studies mentioned above.

E5-8 TOXICITY PROFILE METHAPYRILENE

E5-8.1.1 Chemical Properties and Fate

Methapyrilene is an antihistamine found in over-the-counter sleep medications such as Sominex®and Sleep-eze®.

E5-8.1.2 Bioaccumulation

Methapyrilene is not expected to bioaccumulate at least in mammals. Rats were dosed with methapyrilene by intubation. Approximately 40% and 38% of an administered dose was excreted in urine and feces, respectively, within 24 hours (Kelly et al. 1990). Data were unavailable for plants or other animals.

E5-8.1.3 Aquatic Toxicity

No information was available in the literature reviewed.

E5-8.1.4 Terrestrial Toxicity

No information was available in the literature reviewed.

E5-8.1.5 Mammalian Toxicity

Methapyrilene is a potent rat hepatotoxicant, causing periportal necrosis (Ratra et al. 1998). This study has shown a time- and dose-dependent loss in cell viability in *in vitro* tests. Data suggest that methapyrilene is metabolized by the P450 enzyme CYP2C11 in order to produce toxicity (Ratra et al. 1998). Intoxication leads to dysfunction of the mitochondria, as indicated by mitochondrial swelling and loss of adenosine triphosphate (ATP) within two hours.

Hamsters received 15 mg methapyrilene by gavage two times weekly for 58 weeks (Lijinsky et al. 1983). The total cumulative dose was 15 g/kg, the hamsters exhibited convulsions, but no tumors were reported. Guinea pigs were also studied, and no tumor incidence was reported (Lijinsky et al. 1983). Other studies show that toxicity is not produced in mice at doses up to 1,000 ppm in the diet for 12 weeks (Richardson et al. 1992); whereas a no effect level in the rat is 62.5 ppm in diet. The various data suggest that methapyrilene hepatotoxicity is specific to rats and not observed in other rodents.

Nearly all rats receiving 0.1% methapyrilene in diet developed liver neoplasms; mortality occurred (Lijinsky et al. 1980).

E5-8.1.6 Human Toxicity

There are several reported case histories of suicide by ingestion of methapyrilene containing drugs. One surviving case reported ingesting 100 Sleep-eze tablets (2.5 g methapyrilene), although 1.1 g were recovered by serial lavage (Winek et al. 1977). Thus, an estimated 1.4 g/70 kg body weight, or 20 mg/kg bw, can be estimated as a dose that can be survived.

Tests for hypnotic effects were conducted with humans. A dose of 50 mg methapyrilene was not hypnotic (Teutsch et al. 1975). This equates to a dose of 0.71 mg/kg bw for an average human, which could be used as an RfD.

E5-8.1.7 Recommendations

Since this substance is widely used as a sleep inducing aid, it is recommended that it not be considered further as a contaminant of potential concern for the human health risk assessment. However, if needed for the purposes of quantitative risk analysis, an RfD of 0.71 mg/kg bw/d was estimated for humans. An actual RfD could be higher.

For ecological risks, a rat receptor should be considered since it is the most sensitive species of those tested. The no effect level of 62.5 ppm in diet (or 0.3 mg/kg bw/d for a 200 g rat) should be used as a no effect level for rat receptors, and 1,000 ppm (50 mg/kg bw/d for a 20 g mouse) could be applied to other mammalian receptors.

E5-9 TOXICITY PROFILE
PHENANTHRENE

E5-9.1.1 Chemical Properties and Fate

Sources of polycyclic aromatic hydrocarbon (PAH) compounds include coal derivatives, petroleum products, and forest fires (CEPA 1994). Phenanthrene is considered a low molecular weight PAH compound.

Phenanthrene is similar in structure to anthracene; these two compounds are sterioisomers of one another, and have the same molecular weight and Log Kow (CEPA 1994). The molecular weight is 178.24. The Log Kow is 4.5. The water solubility is 1.25 mg/L at 25°C and differs from anthracene, which is reported as 0.045 mg/L at 25°C (CEPA 1994). The melting point and vapor pressure of phenanthrene also differ from anthracene.

E5-9.1.2 Bioaccumulation

Plant-soil partition coefficients are useful in predicting tissue concentrations in plants that could be used as food for human or ecological receptors. Plant concentrations change over time in response to differences in uptake or loss, but in general uptake is higher earlier in the growing cycle, and slows as the plant matures (Layton et al. 1987). Plant concentration factors (Ksp), which fall within the range estimated by Small (1984) for different classes of organic chemicals, were estimated by the following equation from Topp et al. (1986), which is based on measured uptake values for barley for 14 chemicals and had a r^2 of 0.89:

$$\log Ksp = 5.943 - 2.385 * \log MW$$

The predicted uptake factor or Ksp for plants is 0.57 (Layton et al. 1987).

Measured data for uptake by fish or other aquatic life were unavailable in the literature reviewed. Concentrations in fish were predicted with the following equation from Vieth and Kosian (1983) (Layton et al. 1987). This equation estimates a bioconcentration factor (BCF) for fish, and was developed from data for 122 organic chemicals and has an r^2 of 0.86:

$$\log Kfw = 0.79 * \log Kow - 0.40$$

The predicted concentration factor (Kfw) in fish, based on the log Kow of 4.5, was 1,429. Data indicate that BCFs for fish range between 1,000 and 4,000 (Gerhart et al. 1981).

Estimation of the concentration in beef fat can also be made from regression equations developed by Kenaga (1980) (Layton et al. 1987). These equations relate the Kow and aqueous solubility (S) in mg/L of organic chemicals to their partitioning between diet and fat of cattle. Based on 28 day feeding trials with 23 organic chemicals, the equations are as follows:

$$Log\ Kfd = 0.5 * Log\ Kow - 3.457 \quad (n = 23; r^2 = 0.62)$$

$$Log\ Kfd = -0.495 * Log\ S - 1.476 \quad (n = 23, r^2 = 0.67)$$

The estimated Kfd is 0.06 based on a log Kow of 4.5, and the estimated Kfd based on water solubility was 0.03 based on a water solubility of 1.25 mg/L. These values indicate that grazing mammals should not contain large amounts of phenanthrene.

E5-9.1.3 Aquatic Toxicity

The measured 90-d LC50 for rainbow trout embryos (Oncorhynchus mykiss) was 8 µg/L (CEPA 1994). A 96-h LC50 for juvenile rainbow trout was 375 µg/L, and a 27-d LC50 for embryos was 30 µg/L (CEPA 1994). The 10-day LC50 for two species of copepods exposed to phenanthrene in sediments ranged between 43 to 349 mg/kg dry weight (Lotufo and Fleeger 1997). Reproductive effects including reduced number of offspring, prolonged larval and embryonic stages, and decreased egg hatching success were reported at sediment concentrations as low as 22 mg/kg.

E5-9.1.4 Terrestrial Toxicity

Phenanthrene sorbs to soils and over time weathers or "ages." Soil sorption increases with increased aging, which results in decreased bioavailability (White et al. 1997). Wetting and drying of the soil during the aging process results in further decreased bioavailability as indicated by reduced bacterial mineralization.

E5-9.1.5 Mammalian Toxicity

Data indicate phenanthrene has little or no tumorigenic activity (Wood et al. 1979). In addition, phenanthrene did not demonstrate mutagenicity in tests with mouse liver microsomes under standard test conditions (Beucker et al. 1979). However, substituted phenanthrene derivatives can be mutagenic (Stark et al. 1986).

Orally absorbed rapidly in rats, phenanthrene concentrations in blood plasma peak after 1 hour following dosing (Kadry et al. 1995). Dermal exposure results in maximum blood concentrations 12 hours after dosing. Phenanthrene produced elevated serum AST and gamma-glutamyl transpeptidase (GGTP) 24 hours after dosing in rats (Yoshikawa et al. 1985), suggesting liver injury.

E5-9.1.6 Human Toxicity

No information was available in the literature reviewed.

E5-9.1.7 Recommendations

Because phenanthrene closely resembles anthracene, the oral RfD for anthracene could be used to represent phenanthrene toxicity. However, even though the Kow values are similar between the two compounds, the water solubility is very different. This suggests that toxicological properties also could be different. Therefore, if anthracene values are used to represent phenanthrene, an additional uncertainty factor of at least 10 should be applied in order to conservatively estimate risk.

E5-10 TOXICITY PROFILE SULFATE

E5-10.1.1 Chemical Properties and Fate

Sulfur makes up 0.1% of the earth's crust, and is widely distributed in reduced form (S^{-2}) as metallic sulfides in rock; whereas, sulfates (SO_4^{-2}) occur primarily in sediments and to a much lesser extent in rock (Bodek et al. 1988). The major sulfate minerals are gypsum ($CaSO_4$-$2H_2O$) and anhydrite ($CaSO_4$). Sulfate is the second most abundant anion after Cl- and has a concentration of 2,700 mg/kg. In free or complexed form, sulfate accounts for nearly all of the sulfur in the ocean. Aluminum sulfate and ferrous sulfate are used in drinking water treatment (Bodek et al. 1988).

E5-10.1.2 Bioaccumulation

No information was obtained in the literature reviewed.

E5-10.1.3 Aquatic Toxicity

Freshwater organisms are not adapted to high levels of salts in water. Seawater is considered to be 3.5% total salts, estuarine water is 0.05 to 3.0% salts, and freshwater is <0.05% salts (Rand et al. 1995). Very hard freshwater contains 240 mg/L $CaSO_4$-$2H_2O$ and 240 mg/L $MgSO_4$ (ASTM 1996). Therefore, if sulfate concentrations remain within the normal values for freshwater, toxicity to aquatic life is not expected.

E5-10.1.4 Terrestrial Toxicity

Sulfate is not considered toxic to plants (Bodek et al. 1988). It is an essential nutrient for plants. Soil acidity that often accompanies production of sulfate, such as during the oxidation of pyrite, can be toxic to plants (Bodek et al. 1988).

E5-10.1.5 Mammalian Toxicity

Sulfate is not considered toxic to animals (Bodek et al. 1988).

E5-10.1.6 Human Toxicity

High concentrations of $MgSO_4$ have a laxative effect (Bodek et al. 1988). A sulfate solution of 1,000 mg/L produces vomiting in adults, and lower concentrations have the same effect in children.

The WHO recommended maximum concentration in drinking water is 400 mg/L based on the taste imparted to water when sulfate concentrations exceed 200 to 500 mg/L (Bodek et al. 1988). This indicates that at a consumption rate of 2 L per day, over 800 mg of sulfate can be safely consumed daily in drinking water alone. For a 70 kilogram human, this equates to a safe consumption rate of over 11.4 mg/kg bw/d. Safe doses could be even higher than this, since the drinking water concentration is based on taste and not a health effect. The U.S. Environmental Protection Agency (EPA) drinking water recommendation is 250 mg/L based on taste considerations (Bodek et al. 1988).

E5-10.1.7 Recommendations

Only extremely high sulfate concentrations will be toxic. Therefore, it is recommended that sulfate not be considered further as a contaminant of potential concern.

E5-11 TOXICITY PROFILE TPH DIESEL

E5-11.1.1 Chemical Properties and Fate

Diesel is a complex mixture of petroleum hydrocarbons, the toxicity of which varies according to the mixture properties. Diesel consists of small amounts of polycyclic aromatic hydrocarbons (PAHs), methyl alkanes, and simple alkanes (Heath et al. 1993). The PAHs reported to occur in total petroleum hydrocarbon (TPH)-diesel are benzo(a)pyrene (0.07 ug/kg or 0.7% by weight), methylnapthalene (0.57-0.91%), and napthalene (0.13%) (Heath et al. 1993). The largest alkane fractions (maximum amounts >1%) are summarized below (Heath et al. 1993; CRC 1988):

Chemical	Weight %	Molecular Weight
Decane	0.5–2	142.28
n-Dodecane	0.96–11	170.33
n-Eicosane	0.23–3	282.55
n-Hexadecane	1.2–6	226.44
n-Heptadecane	1.2–6	240.47
n-Nonadecane	0.53–4	268.53
n-Octadecane	0.82–5	254.4
n-Pentadecane	1–7	190.38
n-Tetradecane	1.1–9	198.39
n-Tridecane	1.1–10	184.37
n-Undecane	0.98–9	156.31

Water solubility decreases with increasing carbon number. Data indicate that alkanes with carbon numbers of 10 and higher have water solubilities less than 0.1 mg/L (Nakles et al. 1996). Water saturated with diesel fuel contained less than 0.1 mg/L alkanes, 4.2 mg/L monaromatics, 0.68 mg/L diaromatics, and 0.06 naphthenoaromatics, for a total of 4.9 mg/L diesel fuel (Nakles et al. 1996). Concentrations of napthalene and benzo(a) pyrene in water saturated by diesel are 9E-2 and 6E-9 mg/L, respectively (Nakles et al. 1996).

E5-11.1.2 Bioaccumulation

There was no information available in the literature reviewed.

E5-11.1.3 Aquatic Toxicity

There was no information available in the literature reviewed.

E5-11.1.4 Terrestrial Toxicity

There was no information available in the literature reviewed.

E5-11.1.5 Mammalian Toxicity

Repeated subchronic exposures produce kidney damage in male rats; however, this toxic effect appears to be specific to rats (Nakles et al. 1996). Existing data indicate that neurotoxicity is not likely, and that diesel fuel is not mutagenic nor is it a developmental toxicant (Nakles et al. 1996).

E5-11.1.6 Human Toxicity

Essentially, the largest components of diesel are the C13–C16 aliphatic and the C17+ aliphatic fractions (Nakles et al. 1996). The Total Petroleum Hydrocarbon Criteria Working Group (TPHCWG) suggests using n-nonane as a surrogate for the C9 to C16 fractions, and mineral oil as a surrogate for the C17+ aliphatic frations. Both chemicals have a RfD of 0.6 mg/kg bw/d (Nakles et al. 1996).

An RfD of 0.02 to 0.04 mg/kg bw/d was proposed by Ryer-Powder and Sullivan (1994) for diesel fuel, based on inflammatory and degenerative skin changes in a dermal chronic study (Nakles et al. 1996).

Based on soil volatilization to outdoor air, a Tier 1 soil clean-up goal of 1.22E4 mg/kg was proposed, and a clean-up goal of 2.41E2 mg/kg was proposed for soil vapor intrusion into buildings. To protect groundwater from leaching of TPH, a goal of 1.72E4 was proposed (Nakles et al. 1996). The clean-up goal based on residential risk exceeded soil saturation levels; i.e., the goal was greater than the maximum concentration possible in soil, excluding the presence of free product (Nakles et al. 1996). These goals are extremely conservative, since they assume an infinite source, rely on conservative soil properties, and do not consider any degradation. Thus, they should only be used as a screening tool.

An RfD for diesel can be estimated from information provided by Nakles et al. (1996) by using the surrogate approach. The surrogate approach has been used by the Massachusettes Department of Environmental Protection, and a variation of this approach is also recommended by the TPHCWG. Diesel is broken into its major aliphatic and aromatic fractions, to which each is assigned a surrogate with a known RfD. A toxicity-weighted average is then estimated. The approach is applied as follows:

Fraction	Diesel	Surrogate	RfD (mg/kg bw/d)
C5-C8 aliphatic	0.004	n-hexane	0.06
C9-C16 aliphatic	0.435	n-nonane	0.6
C17+ aliphatic	0.338	Mineral oil	0.6
C6-C8 aromatic	0.001	Toluene	0.2
C9-C16 aromatic	0.105	Naphthalene	0.04
C17+ aromatic	0.117	Pyrene	0.03

RfD = Σ(Fraction*Surrogate RfD)

= 0.47 mg/kg bw/d

E5-11.1.7 Recommendations

The EPA has concluded that diesel fuel should not be considered carcinogenic and that addressing carcinogenicity is more appropriately done by evaluating the individual carcinogenic PAH constituents (Nakles et al. 1996). An RfD of 0.47 mg/kg bw/d should be applied for risk calculations.

E5-12 REFERENCES

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